PTCA in pts with fixed TI defect. Details were as follows:

	Group 1	Group 2	P value
Reference (mm)	2.83 ± 0.43	2.86 ± 0.65	NS
MLD Pre (mm)	0.46 ± 0.44	0.56 ± 0.35	NS
MLD Post (mm)	2.32 ± 0.48	2.23 ± 0.57	NS
MDL F/U (mm)	1.58 ± 0.53	2.10 ± 0.77	<0.05
%DS Pre	83.0 ± 16.2	80.4 ± 11.0	NS
%DS Post	18.2 ± 10.4	22.1 ± 10.8	NS
%DS F/U	45.2 ± 22.2	26.8 ± 18.9	<0.01

Conclusions: Myocardial viability which was confirmed by TI scintigraphy itself appears to provide favorable chronic coronary patency after PTCA procedure.

1164-103 Improved in Hospital Outcome for Patients With Severe Left Ventricular Dysfunction who Undergo Angioplasty

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Patients with a low ejection fraction (<25%) who undergo coronary angioplasty are considered high risk. We tested the hypothesis that the refinement in the technology and the availability of stents should improve the outcome in these patients. Thus we compared 120 patients with a low ejection fraction who underwent coronary angioplasty during 1981–1994 (Group I) to 47 patients who underwent coronary angioplasty during the period of 1995–1996 (Group II). The percent of patients with low ejection fraction who underwent angioplasty relative to the total population for the same time period was higher in group II (1.2% v/s 0.6%, p < 0.0001). Baseline characteristics were similar except for an older population in group II (65 v/s 61 years, p = 0.02). Acute closure occurred more frequently in group I (17.5% v/s 2.1%, p < 0.01). Stents were used more frequently in group II (23.4% v/s 4.2%, p = 0.0004). The use of intra-aortic balloon pump was similar in both groups (2.5% v/s 2.1, p = ns). In hospital coronary artery bypass surgery and death was more common in group I (10.0% v/s 0%, p = 0.02). One year event free survival was 56% in group I and 64% in group II (p = 0.2).

Conclusion: More patients with severe left ventricular dysfunction have been attempted with angioplasty in recent years. The hospital outcome is increasingly more favorable. Long term outcome needs to be evaluated over a longer period of time.

1164-104 Does Periprocedural MI During PTCA Reduce Subsequent Clinical Restenosis?

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Adjunctive treatment of PTCA with abciximab is associated with significant reductions in early events, but the effects of platelet GP IIb/IIIa receptor antagonist therapy on clinical restenosis, as defined by late target vessel revascularization (TVR), remains uncertain as conflicting conclusions have been reached in recent multicenter trials. We sought to determine whether the striking reduction in periprocedural MI afforded by abciximab might confound an accurate assessment of the drug's effects on the need for late TVR. In the placebo-controlled EPIC trial, 2,099 patients with high-risk clinical or angiographic features underwent PTCA or atherectomy and were prospectively followed for 3 years. Within the study population, periprocedural CK elevation was a significant predictor of late mortality. Among patients with elevated CK, however, a paradoxical *decrease* in the need for late TVR was present. This relationship became progressively more profound as the magnitude of CK release increased.

CK ratio	3 yr mortality (%)	3 yr TVR %	(Risk Ratio, 95%CI)
Normal	6.4	29.8	-
<1 × normal	6.2	27.9	(0.92, 0.75–1.14)
<2 ×	6.4	25.4	(0.79, 0.60–1.03)
<3 ×	7.6	24.8	(0.75, 0.55–1.01)
≥5 ×	9.1	20.8	(0.62, 0.42–0.92)
≥10 ×	10.4	16.9	(0.51, 0.29–0.91)

Mechanistically, while it is unlikely that CK elevation prevents vascular renarrowing *per se*, myocardial necrosis impairs the clinical manifestation of restenosis, thereby reducing the need for ischemia-driven TVR.

Conclusion: In the EPIC study, patients with periprocedural MI were less likely to develop clinical restenosis as measured by the need for TVR at 3 years. This novel finding (1) highlights a mechanism for potential discordance between angiographic and clinical measures of restenosis, and (2) has implications for future clinical trials, as therapies which reduce periprocedural MI may be associated with a perceived excess of restenosis when measured by the need for TVR.

1164-105 Is Operator Interpretation or Core lab Analysis More Accurate in Predicting In-hospital Adverse Events After Primary PTCA?

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Although core lab analysis has evolved as the gold standard to judge interventional results, limited data exist regarding whether the core lab or operator is better at predicting adverse events. To address this issue, experienced technicians performed a blinded review of cineangiograms from the PAMI-2 trial. Discrepancies in post PTCA stenosis >20% or TIMI flow grades >1 were adjudicated by a third angiographer. Operator and core lab interpretations were individually placed into a multivariate model to predict in-hospital recurrent MI, or ischemia. Primary PTCA was performed in 986 patients, of whom 1.9% had reMI and 10.5% had ischemia. The only correlate of ischemia or reMI was residual stenosis >30% or the presence of a dissection; TIMI flow and thrombus grade were not predictive.

	Operator		Core		
Recurrent MI	P value	CI*	P value	CI*	
Stenosis	0.0015	0.60	0.0007	0.28	
Dissection	0.0007	0.60	NS	-	
Ischemia					
Stenosis		0.004	0.25	0.006	0.16
Dissection	0.01	0.25	NS	-	

*Higher correlation index (Somers' D) = better predictive ability

Therefore, operator interpretation appears to be superior to core lab analysis in predicting in-hospital recurrent ischemia or reinfarction. Whether this is due to use of fluoroscopic findings which were not recorded on cine, acute use of digital imaging or greater operator experience deserves further study.

1164-106 In-Vivo Identification and Characterization of Angioplasty-Induced Injury Using Vascular Acoustic Emissions

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Background: We have previously demonstrated in-vitro that atherosclerotic (ATH) arterial tissue emits sound as it experiences mechanical disruption.

Methods: To further characterize acoustic emission (AE) patterns and to identify if AE occur in-vivo at the time of balloon angioplasty (BA), 24 human arterial specimens obtained post-mortem and 8 ATH and control arteries in-vivo (miniswine) were evaluated. Arteries were subjected to BA with simultaneous recording of AE using a balloon-mounted piezoelectric transducer. The recorded signals were analyzed in the time domain using wavelet techniques and in the frequency domain using Fourier techniques, and then characterized according to the type of induced trauma using Kohonen neural network classification.

Results: Vascular AE were demonstrated to occur between 30 and 5000 Hz. Using spectrograms with Kohonen classification, the sounds were characterized as noiseless, plaque fracture and tearing. The in-vivo data indicate fewer extraneous signals and the same AE patterns as observed in the in-vitro studies. Tearing is associated with relatively long (0.4–1.0s) 'zipping' AE, balloon opening with shorter and lower frequency AE, and fracture with even shorter (0.1 s) and 'crisper' AE.

Conclusion: We have demonstrated that vascular acoustic emissions occur following atheroma injury both in-vitro and in-vivo. Waveform analysis helps to further distinguish normal from pathologic vascular injury. These data support the concept of using acoustic emissions as a means of monitoring and optimizing balloon angioplasty.